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L40: Entry 1 of 2

File: USPT

Oct 29, 2002

DOCUMENT-IDENTIFIER: US 6472154 B1
 TITLE: Polymorphic repeats in human genes

Detailed Description Paragraph Table (170):

L26953 3'UTR 4.16 2797 CAAAAA L27560 UNKNOWN 6 3438 GTT L27560 UNKNOWN 14 3658 A L27745
 CDS 7 1067 AG L30117 UNKNOWN 79 652 T L31881 3'UTR 4.59 1481 CCCAG L32832 CDS 2.93 2092
 GAGGAGGAGGAAGAAA (SEQ ID NO:254) L32832 CDS 10 5866 CAA L32832 CDS 7.66 10261 CAG L32832
 CDS 7 2985 GGC L33075 3'UTR 16 6723 A L33243 3'UTR 9 13947 TG L33477 3'UTR 10 3557 CA
 L34357 CDS 6 590 GGC L34408 UNKNOWN 15 706 A L35592 UNKNOWN 14 1577 AC L36140 5'UTR 24
 1 T L36642 3'UTR 4 4333 CAAAAA L36983 3'UTR 5.66 3368 CTC L37112 CDS 3.5 1257 CCTCAG
 L37198 UNKNOWN 23 7 A L38707 CDS 3.83 146 CCGGGG L38951 3'UTR 31 3653 A L38951 3'UTR 13
 3736 T L38961 3'UTR 22 2283 T L39064 CDS 8.33 1504 AGC L39833 3'UTR 13 1809 A L39833
 3'UTR 13 2713 T L40377 5'UTR 6.66 33 GCG L40377 5'UTR 6.33 51 GCA L40392 3'UTR 14 2209
 T L40992 CDS 6 18 CAG L40992 BORDER 5.66 0 CAG L41690 CDS 6 620 GCC L41887 3'UTR 14
 2046 A L41919 CDS 8 440 GGC L42025 5'UTR 5.66 1 GGC L42243 3'UTR 7 3934 GT L42243 3'UTR
 16 2217 T L42243 3'UTR 15 1137 T L42243 3'UTR 12 2874 A L44505 UNKNOWN 13 309 A L46353
 5'UTR 2.8 2094 CACACTCACA (SEQ ID NO:255) L46353 5'UTR 19 2401 TC L46353 5'UTR 9.5 2381
 TC L46353 5'UTR 6.5 1394 TG L46353 5'UTR 13 264 A L46353 3'UTR 5 3378 TGGGG L48796
 UNKNOWN 15 147 A L49169 3'UTR 6 1661 GAG L49169 3'UTR 6.5 2689 CT L49380 CDS 6 1771 GCC
 L76702 CDS 4.33 301 CAGCCC L76703 3'UTR 12 2219 A L77864 CDS 5.66 571 GAG L78833 3'UTR
 18 6465 A M10901 3'UTR 18 3217 A M11220 3'UTR 5.5 693 TATT M11353 3'UTR 16 817 A M11722
 5'UTR 14 216 G M12783 UNKNOWN 3.85 296 CGCAGCT M12783 UNKNOWN 7.5 3612 AC M12783
 UNKNOWN 16 238 A M13232 3'UTR 6.5 1889 CA M13452 3'UTR 8.5 2030 GA M13452 3'UTR 15 1745
 A M13903 CDS 5 528 GAGCAGCAGGGAGGGCAGCTGGAGCTCCCA (SEQ ID NO:256) M13903 CDS 3.43 679
 AGCAGCAGGAGGGCAGCTGGAGCTCTG (SEQ ID NO:257) M14058 3'UTR 12 2221 A M14083 3'UTR 7
 2667 AT M14170 CDS 7.66 30 TGC M14219 3'UTR 13 1660 T M14630 UNKNOWN 12 538 A M14648
 3'UTR 10.66 3535 TTG M14745 3'UTR 7.5 897 AC M14764 5'UTR 3.6 36 AGCGC M14764 3'UTR 7
 2444 CA M15169 UNKNOWN 13 2852 C M15353 3'UTR 18 847 T M16276 UNKNOWN 12 1368 A M16505
 UNKNOWN 6.5 2611 AC M16505 UNKNOWN 18 3645 A M16801 CDS 3.83 2295 CCCCCA M16937 3'UTR
 2.7 865 AAACAAA (SEQ ID NO:258) M16938 5'UTR 7 172 TG M16965 CDS 2.94 115
 TACCTTGTGGAAGACG (SEQ ID NO:259) M16965 CDS 2.5 591 CTGGAAGACATGGATTTT (SEQ ID
 NO:260) M18533 UNKNOWN 5.25 12297 TTGA M18533 UNKNOWN 8.5 11725 AC M18728 UNKNOWN 12
 2266 A M19154 3'UTR 7.33 2117 ACA M19961 5'UTR 13 0 T M20681 UNKNOWN 13 2233 T M20681
 UNKNOWN 13 3705 A M21305 CDS 6.4 56 TGGAA M21305 BORDER 3.8 0 ATGGA M21574 3'UTR 14
 4503 T M23114 3'UTR 3.8 3839 CACCC M23263 CDS 20.33 1884 GGC M23263 CDS 17 701 GCA
 M24069 CDS 7.66 229 CCA M24283 3'UTR 9 2742 GT M24486 3'UTR 16 2350 T M24902 3'UTR 12.5
 2338 AATA M25667 3'UTR 9 1153 CT M25667 3'UTR 17 973 A M28170 3'UTR 17 1816 GT M28713
 5'UTR 6.66 253 CGG M29053 3'UTR 3.8 1579 AAAAT M29204 UNKNOWN 21 288 A M29873 3'UTR 6.5
 1687 TA M29874 3'UTR 7 1688 AT M29874 3'UTR 15 2510 T M30448 3'UTR 26 887 A M31165
 BORDER 12 900 A M31523 3'UTR 12 2316 T M31525 3'UTR 6.5 1037 AC M31682 3'UTR 8 1601 AG
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 1430 T M32315 3'UTR 7.75 1814 TTTG M32315 3'UTR 6.5 3589 TG M32315 3'UTR 12 2368 A
 M34041 CDS 6.66 903 GAG M34309 5'UTR 6.5 4 CA M34539 3'UTR 12 1120 T M35531 3'UTR 6.25
 2068 TTAT M35531 3'UTR 18 1752 T M35663 3'UTR 18 2056 T M36089 UNKNOWN 17 2462 AC
 M36542 3'UTR 18 1886 A M36711 3'UTR 6.66 1494 GCC M36820 3'UTR 6.25 540 TATT M36860 CDS
 3.66 950 CAGCTG M37981 CDS 6.33 113 GCT M54915 5'UTR 3.8 246 CAGCA M54915 5'UTR 9 45
 GCA M54915 3'UTR 5.25 2191 TATT M54927 3'UTR 14 2476 A M55047 3'UTR 22.5 2095 GT M55053
 3'UTR 12 1915 T M55172 CDS 7.96 3238 CTGCCCTGGAGTAGAGGACATCAGCAGGGCTTCCTCTG
 GAGAAGTTCTAGAGACCG (SEQ ID NO:261) M55172 CDS 5.52 2979
 GGGCTTCCTCTGGAGAAGTTCTAGAGACCACTGCCCT GGAGTAGAGGACATCAGC (SEQ ID NO:262) M55172 CDS 5
 3636 GCTGCCCTGGAGTAGAGGACATCAGCAGGGCTTCCTCT GGAGAAGTTCTAGAGACT (SEQ ID NO:263) M55422
 5'UTR 5.66 944 TTG M55542 3'UTR 14 2006 A M55593 5'UTR 7.33 87 GCG M55630 UNKNOWN 22
 126 GT M55630 UNKNOWN 12 1538 A M55654 CDS 18.66 466 CAG M55654 CDS 9.66 430 CAG M55654
 3'UTR 13 1297 T M55683 3'UTR 12 1203 TG M57627 3'UTR 3.5 1457 AAAAT M58583 3'UTR 5.66
 1354 TTG M59305 5'UTR 4.16 129 CTTTT M59465 3'UTR 12 3986 A M59499 3'UTR 2.91 2093

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GGAGGAAGAGAAAAGGGCAGCAGAGGAGAGGCAGAGGAT AAA (SEQ ID NO:266) M83738 3'UTR 9 2798 TG M83751 CDS 8 256 GGA M84424 3'UTR 8.5 1341 AC M84739 5'UTR 14 25 C M85169 3'UTR 12 1712 T M85590 UNKNOWN 17 0 T M85670 UNKNOWN 13 227 A M86400 3'UTR 18 1632 T M86406 5'UTR 3.57 71 CGCCCCG M86406 3'UTR 6.5 2952 GA M86699 5'UTR 15 8 T M87503 CDS 7 574 AGC M87503 3'UTR 13 1400 T M88108 3'UTR 12 1749 A M88282 3'UTR 7.75 4728 AAAG M88282 3'UTR 16 4715 A M89796 3'UTR 23 2068 A M89796 3'UTR 14 1205 T M90391 CDS 6.66 694 CCT M90656 5'UTR 9.66 55 GGA M91463 3'UTR 23 2202 T

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L39	6303301[pn] or 6020135[pn]	2	L39
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L29	L28 or l25	78	L29
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L27	(brain or cerebral or (choroid adj plexus) or cerebellum or hypothalamic) same (tumor\$1 or tumour\$1 or cancer\$1 or cancerous or neoplas\$3 or carcinoma\$1 or teratoma\$1 or papilloma\$1) glioma\$1 or glioblastoma\$1 or medulloblastoma\$1 or neurocytoma\$1 or pinealoma\$1 or astrocytoma\$1 or ependymoma\$1 or craniopharyngioma\$1	4963	L27
L26	FGFR3 or (FGF adj R3) or (fibroblast adj growth adj factor adj receptor)	1273	L26
<i>DB=USPT; PLUR=NO; OP=ADJ</i>			
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L16	L15[ti,ab]	105	L16
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L12	brain same (tumor\$1 or tumour\$1 or cancer\$1 or cancerous or neoplas\$3 or carcinoma\$1 or teratoma\$1 or papilloma\$1)	8008	L12
L11	L10[ti,ab]	0	L11
L10	cek2 or (cek adj 2) or FGFR3 or (FGF adj R3) or (fibroblast adj growth adj factor adj receptor adj (3 or III))	59	L10
L9	cek2 same (tumor\$1 or tumour\$1 or cancer\$1 or cancerous or neoplas\$3 or carcinoma\$1 or teratoma\$1 or papilloma\$1 or glioma\$1 or glioblastoma\$1 or medulloblastoma\$1)	3	L9
L8	L7 and @ad<20010131	3	L8
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L6	cek or (cek adj (2 or II))	62	L6
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L4	L2 and @prad<20010131	6	L4
L3	L2 and @ad<20010131	14	L3
L2	L1 same (tumor\$1 or tumour\$1 or cancer\$1 or cancerous or neoplas\$3 or carcinoma\$1 or teratoma\$1 or papilloma\$1 or glioma\$1 or glioblastoma\$1 or medulloblastoma\$1)	16	L2
L1	FGFR3 or (FGF adj R3) or (fibroblast adj growth adj factor adj receptor adj (3 or III))	52	L1

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LS5: Entry 11 of 16

File: USPT

Apr 10, 2001

DOCUMENT-IDENTIFIER: US 6214795 B1

TITLE: Peptide compounds useful for modulating FGF receptor activity

DATE FILED (1):19961112Brief Summary Text (4):

The FGFs mediate their effects by binding to high affinity cell surface receptors (reviewed in Johnson and Williams (1992) *Adv. Cancer Res.* 60:1-41). Four distinct FGF receptors have been identified: FGFR1 (also known was Flg, bFGFR, Cek1 or N-bFGFR) (Lee et al. (1989) *Science* 245:57-60; Dionne et al. (1990) *EMBO J.* 9:2685-2692; Johnson et al. (1990) *Mol. Cell. Biol.* 10:4728-4736; Eisemann et al. (1991) *Oncogene* 6:1195-1202; Hou et al. (1991) *Science* 251:665-668), FGFR2 (also known as Bek, Cek3, K-sam, TK14, TK25 or KGFR) (Dionne et al. (1990) *EMBO J.* 9:2685-2692; Hattori et al. (1990) *Proc. Natl. Acad. Sci. USA* 87:5983-5987; Miki et al. (1991) *Science* 251:72-75; Saiki et al. (1988) *Science* 239:487-491; Pasquale (1990) *Proc. Natl. Acad. Sci. USA* 87:5812-5816; Houssaint et al. (1990) *Proc. Natl. Acad. Sci. USA* 87:8180-8184; Champion-Arnaud et al. (1991) *Oncogene* 6:979-987; Crumley et al. (1991) *Oncogene* 6:2255-2262; Raz et al. (1991) *Oncogene* 6:753-760; Sato et al. (1991) *Oncogene* 6:1279-1283), FGFR3 (also known as Cek2) (Keegan et al. (1991) *Proc. Natl. Acad. Sci. USA* 88:1095-1099) and FGFR4 (Partanen et al. (1991) *EMBO J.* 10:1347-1354).

6268391 + 61112
6316479

Isolation of an additional member of the fibroblast growth factor receptor family, FGFR-3.

Keegan K, Johnson DE, Williams LT, Hayman MJ.

Department of Microbiology, State University of New York, Stony Brook 11794.

The fibroblast growth factors are a family of polypeptide growth factors involved in a variety of activities including mitogenesis, angiogenesis, and wound healing.

Fibroblast growth factor receptors (FGFRs) have previously been identified in chicken, mouse, and human and have been shown to contain an extracellular domain

with either two or three immunoglobulin-like domains, a transmembrane domain, and a cytoplasmic tyrosine kinase domain. We have isolated a human cDNA for

another tyrosine kinase receptor that is highly homologous to the previously described FGFR. Expression of this receptor cDNA in COS cells directs the expression

of a 125-kDa glycoprotein. We demonstrate that this cDNA encodes a biologically active receptor by showing that human acidic and basic fibroblast growth factors

activate this receptor as measured by $^{45}\text{Ca}^{2+}$ efflux assays. These data establish the existence of an additional member of the FGFR family that we have named

FGFR-3.

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L5: Entry 15 of 16

File: USPT

Jul 21, 1998

DOCUMENT-IDENTIFIER: US 5783683 A

TITLE: Antisense oligonucleotides which reduce expression of the FGFR1 gene

DATE FILED (1):19950110Drawing Description Text (10):

FIG. 9 shows an RT-PCR Southern Blot of FGFR1, FGFR3, and FGFR4 demonstrating the selective reduction of FGFR1 mRNA following treatment of glioblastoma cells with the antisense molecules of the invention.

Detailed Description Text (122):

mRNA was analyzed as described above with the exception that both FGFR1, FGFR3 and FGFR4 mRNA were studied in this particular work. SNB-19 glioblastoma cells were plated at 1.times.10.^{sup.5} cells per 100 mm dish in serum-supplemented medium. Eighteen hours later the cells were converted to serum-free medium containing FGFR1.alpha. antisense oligonucleotide (R1AS.alpha., 30 .mu.m) or FGFR1.alpha. antisense reverse control oligonucleotide (R1.alpha.RC, 30 .mu.m). Non-treated cells (NT) were run as a control. Cells were treated for three consecutive days with oligonucleotide. Cells were scraped on day 7 and mRNA and cDNA were purified and synthesized respectively. Using cDNA from each of the three different treatments, PCR was used to amplify cDNA for FGFR1, FGFR3, and FGFR4 receptors. SNB-19 cells do not produce FGFR2.



"Brain Neoplasms" [MESH] AND "Receptors, Fibroblast Growth Factor" [Search]

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Brain Tumor Pathol. 2002;19(1):23-9.

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Autocrine signaling through Ras regulates cell survival activity in human glioma cells: potential cross-talk between Ras and the phosphatidylin 3-kinase-Akt pathway.

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J Neuropathol Exp Neurol. 2002 Nov;61(11):975-83.
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Fibroblast growth factor receptor 4 is a target for the zinc-finger transcription factor Ikaros in the pituitary.

Yu S, Asa SL, Ezzat S.
Mol Endocrinol. 2002 May;16(5):1069-78.
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Fibroblast growth factor receptor (FGFR) 4 correlated with the malignancy of human astrocytomas.

Yamada SM, Yamada S, Hayashi Y, Takahashi H, Teramoto A, Matsumoto T.
Neurol Res. 2002 Apr;24(3):244-8.
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Antitumor activity of fibroblast growth factors (FGFs) for medulloblastoma may correlate with FGF receptor expression and tumor variant.

Duplan SM, Theoret Y, Kenigsberg RL.
Clin Cancer Res. 2002 Jan;8(1):246-57.

PMID: 11801566 [PubMed - indexed for MEDLINE]

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Targeted expression of a human pituitary tumor-derived isoform of F receptor-4 recapitulates pituitary tumorigenesis.

Ezzat S, Zheng L, Zhu XF, Wu GE, Asa SL.

J Clin Invest. 2002 Jan;109(1):69-78.

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Pituitary adenomas in man and mouse: oncogenic potential of a truncated fibroblast growth factor receptor 4.

Low MJ.

J Clin Invest. 2002 Jan;109(1):15-6. No Abstract Available.

PMID: 11781345 [PubMed - indexed for MEDLINE]

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Histological and genetic diagnosis of gliomatosis cerebri: case report.

Yamada SM, Hayashi Y, Takahashi H, Teramoto A, Matsumoto K, Yamada T.

J Neurooncol. 2001 May;52(3):237-40.

PMID: 11519853 [PubMed - indexed for MEDLINE]

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Inhibition of fibroblast growth factor/fibroblast growth factor receptor activity in glioma cells impedes tumor growth by both angiogenesis-dependent and -independent mechanisms.

Auguste P, Gursel DB, Lemiere S, Reimers D, Cuevas P, Carceller F, Di Stefano JP, Bikfalvi A.

Cancer Res. 2001 Feb 15;61(4):1717-26.

PMID: 11245488 [PubMed - indexed for MEDLINE]

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Correlation of bFGF, FGFR-1 and VEGF expression with vascularity and malignancy of human astrocytomas.

Bian XW, Du LL, Shi JQ, Cheng YS, Liu FX.

Anal Quant Cytol Histol. 2000 Jun;22(3):267-74.

PMID: 10872046 [PubMed - indexed for MEDLINE]

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Suppression of glioblastoma cell growth following antisense oligonucleotide-mediated inhibition of fibroblast growth factor receptor 1 expression.

Yamada SM, Yamaguchi F, Brown R, Berger MS, Morrison RS.

Glia. 1999 Oct;28(1):66-76.

PMID: 10498824 [PubMed - indexed for MEDLINE]

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[Inhibition of fibroblast growth factor receptor 1 expression in human glioblastoma cell contributes to the cell growth suppression]

Yamada SM, Yamaguchi F, Morrison RS, Takahashi H, Teramoto A.

No To Shinkei. 1998 Dec;50(12):1101-5. Japanese.

PMID: 9989355 [PubMed - indexed for MEDLINE]

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13 *Altered expression of fibroblast growth factor receptors in human pituitary adenomas.*

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J Clin Endocrinol Metab. 1997 Apr;82(4):1160-6.

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Growth Factors. 1995;12(1):49-55.

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J Neurosurg. 1995 Jan;82(1):83-90.

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Kaipainen A, Vlaykova T, Hatva E, Bohling T, Jekunen A, Pyrhonen S, A K.

Cancer Res. 1994 Dec 15;54(24):6571-7.

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Neurosci Lett. 1994 Apr 25;171(1-2):192-6.

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19 *Expression of fibroblast growth factor receptor-1 in human glioma and meningioma tissues.*

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Neurosurgery. 1994 Feb;34(2):221-5; discussion 225-6.

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Yamaguchi F, Saya H, Bruner JM, Morrison RS.

Proc Natl Acad Sci U S A. 1994 Jan 18;91(2):484-8.

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M64347. Human novel growth...[gi:182564]

Links

LOCUS HUMFGFLR 3829 bp mRNA linear PRI 31-DEC-1994

DEFINITION Human novel growth factor receptor mRNA, 3' cds.

ACCESSION M64347

VERSION M64347.1 GI:182564

KEYWORDS growth factor receptor.

SOURCE Homo sapiens (human)

ORGANISM Homo sapiens

Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Primates; Catarrhini; Hominidae; Homo.

REFERENCE 1 (bases 1 to 3829)

AUTHORS Thompson, L.M., Plummer, S., Schalling, M., Altherr, M.R., Gusella, J.F., Housman, D.E. and Wasmuth, J.J.

TITLE A gene encoding a fibroblast growth factor receptor isolated from the Huntington disease gene region of human chromosome 4

JOURNAL Genomics 11 (4), 1133-1142 (1991)

MEDLINE 92147110

PUBMED 1664411

COMMENT Original source text: Homo sapiens cDNA to mRNA.

FEATURES Location/Qualifiers

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/map="4p16.3 D4599"

gene 1..3829

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NM_000142. Homo sapiens fibr...[gi:13112046]

Links

LOCUS FGFR3 4093 bp mRNA linear PRI 21-FEB-2001
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 ORGANISM Homo sapiens
 Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Primates; Catarrhini; Hominidae; Homo.
 REFERENCE 1 (bases 1 to 4093)
 AUTHORS Partanen,J., Makela,T.P., Alitalo,R., Lehvaslaiho,H. and Alitalo,K.
 TITLE Putative tyrosine kinases expressed in K-562 human leukemia cells
 JOURNAL Proc. Natl. Acad. Sci. U.S.A. 87 (22), 8913-8917 (1990)
 MEDLINE 91062389
 PUBMED 2247464
 REFERENCE 2 (bases 1 to 4093)
 AUTHORS Keegan,K., Johnson,D.E., Williams,L.T. and Hayman,M.J.
 TITLE Isolation of an additional member of the fibroblast growth factor receptor family, FGFR-3
 JOURNAL Proc. Natl. Acad. Sci. U.S.A. 88 (4), 1095-1099 (1991)
 MEDLINE 91142118
 PUBMED 1847508
 REFERENCE 3 (bases 1 to 4093)
 AUTHORS Thompson,L.M., Plummer,S., Schalling,M., Altherr,M.R., Gusella,J.F., Housman,D.E. and Wasmuth,J.J.
 TITLE A gene encoding a fibroblast growth factor receptor isolated from the Huntington disease gene region of human chromosome 4
 JOURNAL Genomics 11 (4), 1133-1142 (1991)
 MEDLINE 92147110
 PUBMED 1664411
 REFERENCE 4 (bases 1 to 4093)
 AUTHORS Velinov,M., Slaugenhaupt,S.A., Stoilov,I., Scott,C.I. Jr., Gusella,J.F. and Tsipouras,P.
 TITLE The gene for achondroplasia maps to the telomeric region of chromosome 4p
 JOURNAL Nat. Genet. 6 (3), 314-317 (1994)
 MEDLINE 94282083
 PUBMED 8012397
 REFERENCE 5 (bases 1 to 4093)
 AUTHORS Le Merrer,M., Rousseau,F., Legeai-Mallet,L., Landais,J.C., Pelet,A., Bonaventure,J., Sanak,M., Weissenbach,J., Stoll,C., Munich,A. et al.
 TITLE A gene for achondroplasia-hypochondroplasia maps to chromosome 4p
 JOURNAL Nat. Genet. 6 (3), 318-321 (1994)

MEDLINE 94282084
 PUBMED 8012398
 REFERENCE 6 (bases 1 to 4093)
 AUTHORS Francomano, C.A., Ortiz de Luna, R.I., Hefferon, T.W., Bellus, G.A.,
 Turner, C.E., Taylor, E., Meyers, D.A., Blanton, S.H., Murray, J.C.,
 McIntosh, I. et al.
 TITLE Localization of the achondroplasia gene to the distal 2.5 Mb of
 human chromosome 4p
 JOURNAL Hum. Mol. Genet. 3 (5), 787-792 (1994)
 MEDLINE 94362678
 PUBMED 8081365
 REFERENCE 7 (bases 1 to 4093)
 AUTHORS Perez-Castro, A.V., Wilson, J. and Altherr, M.R.
 TITLE Genomic organization of the human fibroblast growth factor receptor
 3 (FGFR3) gene and comparative sequence analysis with the mouse
 Fgfr3 gene
 JOURNAL Genomics 41 (1), 10-16 (1997)
 MEDLINE 97271550
 PUBMED 9126476
 REFERENCE 8 (bases 1 to 4093)
 AUTHORS Passos-Bueno, M.R., Wilcox, W.R., Jabs, E.W., Sertie, A.L., Alonso, L.G.
 and Kitoh, H.
 TITLE Clinical spectrum of fibroblast growth factor receptor mutations
 JOURNAL Hum. Mutat. 14 (2), 115-125 (1999)
 MEDLINE 99355711
 PUBMED 10425034
 COMMENT REVIEWED REFSEQ: This record has been curated by NCBI staff. The
 reference sequence was derived from M58051.1 and M64347.1.
 On Feb 23, 2001 this sequence version replaced gi:4503710.
 Summary: The protein encoded by this gene is a member of the
 fibroblast growth factor receptor family, where amino acid sequence
 is highly conserved between members and throughout evolution. FGFR
 family members differ from one another in their ligand affinities
 and tissue distribution. A full-length representative protein would
 consist of an extracellular region, composed of three
 immunoglobulin-like domains, a single hydrophobic membrane-spanning
 segment and a cytoplasmic tyrosine kinase domain. The extracellular
 portion of the protein interacts with fibroblast growth factors,
 setting in motion a cascade of downstream signals, ultimately
 influencing mitogenesis and differentiation. This particular family
 member binds acidic and basic fibroblast growth hormone and plays a
 role in bone development and maintenance. Mutations in this gene
 lead to craniosynostosis and multiple types of skeletal dysplasia.
 Alternative splicing occurs and additional variants have been
 described, including those utilizing alternate exon 8 rather than
 9, but their full-length nature has not been determined.
 Transcript Variant: This variant (1) is missing alternatively
 spliced exon 8 but utilizes alternatively spliced exon 9, resulting
 in isoform (1) with the IIIc-type C-terminal half of the IgIII
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COMPLETENESS: complete on the 3' end.

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Links

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 REFERENCE 1 (bases 1 to 3757)
 AUTHORS Partanen,J., Makela,T.P., Alitalo,R., Lehvastalo,H. and Alitalo,K.
 TITLE Putative tyrosine kinases expressed in K-562 human leukemia cells
 JOURNAL Proc. Natl. Acad. Sci. U.S.A. 87 (22), 8913-8917 (1990)
 MEDLINE 91062389
 PUBMED 2247464
 REFERENCE 2 (bases 1 to 3757)
 AUTHORS Keegan,K., Johnson,D.E., Williams,L.T. and Hayman,M.J.
 TITLE Isolation of an additional member of the fibroblast growth factor receptor family, FGFR-3
 JOURNAL Proc. Natl. Acad. Sci. U.S.A. 88 (4), 1095-1099 (1991)
 MEDLINE 91142118
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 REFERENCE 3 (bases 1 to 3757)
 AUTHORS Thompson,L.M., Plummer,S., Schalling,M., Altherr,M.R., Gusella,J.F., Housman,D.E. and Wasmuth,J.J.
 TITLE A gene encoding a fibroblast growth factor receptor isolated from the Huntington disease gene region of human chromosome 4
 JOURNAL Genomics 11 (4), 1133-1142 (1991)
 MEDLINE 92147110
 PUBMED 1664411
 REFERENCE 4 (bases 1 to 3757)
 AUTHORS Velinov,M., Slaugenhaupt,S.A., Stoilov,I., Scott,C.I. Jr., Gusella,J.F. and Tsipouras,P.
 TITLE The gene for achondroplasia maps to the telomeric region of chromosome 4p
 JOURNAL Nat. Genet. 6 (3), 314-317 (1994)
 MEDLINE 94282083
 PUBMED 8012397
 REFERENCE 5 (bases 1 to 3757)
 AUTHORS Le Merrer,M., Rousseau,F., Legeai-Mallet,L., Landais,J.C., Pelet,A., Bonaventure,J., Sanak,M., Weissenbach,J., Stoll,C., Munnich,A. et al.
 TITLE A gene for achondroplasia-hypochondroplasia maps to chromosome 4p
 JOURNAL Nat. Genet. 6 (3), 318-321 (1994)

MEDLINE 94282084
 PUBMED 8012398
 REFERENCE 6 (bases 1 to 3757)
 AUTHORS Francomano,C.A., Ortiz de Luna,R.I., Hefferon,T.W., Bellus,G.A., Turner,C.E., Taylor,E., Meyers,D.A., Blanton,S.H., Murray,J.C., McIntosh,I. et al.
 TITLE Localization of the achondroplasia gene to the distal 2.5 Mb of human chromosome 4p
 JOURNAL Hum. Mol. Genet. 3 (5), 787-792 (1994)
 MEDLINE 94362678
 PUBMED 8081365
 REFERENCE 7 (bases 1 to 3757)
 AUTHORS Perez-Castro,A.V., Wilson,J. and Altherr,M.R.
 TITLE Genomic organization of the human fibroblast growth factor receptor 3 (FGFR3) gene and comparative sequence analysis with the mouse Fgfr3 gene
 JOURNAL Genomics 41 (1), 10-16 (1997)
 MEDLINE 97271550
 PUBMED 9126476
 REFERENCE 8 (bases 1 to 3757)
 AUTHORS Passos-Bueno,M.R., Wilcox,W.R., Jabs,E.W., Sertie,A.L., Alonso,L.G. and Kitoh,H.
 TITLE Clinical spectrum of fibroblast growth factor receptor mutations
 JOURNAL Hum. Mutat. 14 (2), 115-125 (1999)
 MEDLINE 99355711
 PUBMED 10425034
 COMMENT REVIEWED REFSEQ: This record has been curated by NCBI staff. The reference sequence was derived from AF245114.1 and M64347.1.
 Summary: The protein encoded by this gene is a member of the fibroblast growth factor receptor family, where amino acid sequence is highly conserved between members and throughout evolution. FGFR family members differ from one another in their ligand affinities and tissue distribution. A full-length representative protein would consist of an extracellular region, composed of three immunoglobulin-like domains, a single hydrophobic membrane-spanning segment and a cytoplasmic tyrosine kinase domain. The extracellular portion of the protein interacts with fibroblast growth factors, setting in motion a cascade of downstream signals, ultimately influencing mitogenesis and differentiation. This particular family member binds acidic and basic fibroblast growth hormone and plays a role in bone development and maintenance. Mutations in this gene lead to craniosynostosis and multiple types of skeletal dysplasia. Alternative splicing occurs and additional variants have been described, including those utilizing alternate exon 8 rather than 9, but their full-length nature has not been determined.
 Transcript Variant: This variant (2) does not contain alternatively spliced exons 8 or 9, resulting in a loss of the C-terminal half of the IgIII domain. In addition, this variant is missing alternatively spliced exon 10 which encodes the transmembrane region, suggesting a soluble receptor.

COMPLETENESS: complete on the 3' end.

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M58051. Human fibroblast ...[gi:182568]

Links

LOCUS HUMFGFR3 2520 bp mRNA linear PRI 08-NOV-1994
DEFINITION Human fibroblast growth factor receptor (FGFR3) mRNA, complete cds.
ACCESSION M58051
VERSION M58051.1 GI:182568
KEYWORDS fibroblast growth factor receptor.
SOURCE Homo sapiens (human)
ORGANISM Homo sapiens
Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi;
Mammalia; Eutheria; Primates; Catarrhini; Hominidae; Homo.
REFERENCE 1 (bases 1 to 2520)
AUTHORS Keegan, K., Johnson, D.E., Williams, L.T. and Hayman, M.J.
TITLE Isolation of an additional member of the fibroblast growth factor
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JOURNAL Proc. Natl. Acad. Sci. U.S.A. 88 (4), 1095-1099 (1991)
MEDLINE 91142118
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gi|182568|gb|M58051.1|HUMFGFR3 Human fibroblast growth factor receptor (FGFR3)
mRNA, complete cds
Length = 2520

Score = 4391 bits (2215), Expect = 0.0

Identities = 2244/2256 (99%)

Strand = Plus / Plus

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AF487554. *Homo sapiens* fibr...[gi:20452379]

Links

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 VERSION AF487554.1 GI:20452379
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 ORGANISM *Homo sapiens*
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 AUTHORS Lind,D.L. and Cox,D.R.
 TITLE Fibroblast growth factor receptor 3 (FGFR3) genomic sequence
 JOURNAL Unpublished
 REFERENCE 2 (bases 1 to 16976)
 AUTHORS Lind,D.L. and Cox,D.R.
 TITLE Direct Submission
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AUTHORS Terada,M., Shimizu,A., Sato,N., Miyakaze,S.I., Katayama,H. and Kurokawa-Seo,M.
TITLE Fibroblast growth factor receptor 3 lacking the Ig IIIb and transmembrane domains secreted from human squamous cell carcinoma DJM-1 binds to FGFs
JOURNAL Mol. Cell Biol. Res. Commun. 4 (6), 365-373 (2001)
MEDLINE 21561228
PUBMED 11703096
REFERENCE 2 (bases 1 to 2184)
AUTHORS Terada,M., Shimizu,A. and Seo,M.
TITLE Secretion and dimerization of the FGFR3 isoform, resulting from alternative splicing, that is expressed in human malignant trichilemmal cyst cell
JOURNAL Unpublished
REFERENCE 3 (bases 1 to 2184)
AUTHORS Terada,M., Shimizu,A. and Seo,M.
TITLE Direct Submission
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Sbjct: 14045 ccctccaaggctaaaagggtttaatagttggaggtgattccagtgaagatatttattt 14104

```
Query: 3272  gctttgtccttttcaggagaatttagattctataaggattttctttaggagattt 3331
          ||||||| ||||||| ||||||| ||||||| ||||||| ||||||| ||||||| ||||||| |||||
Sbjct: 14105 cctttgtccttttcaggagaatttagattctataaggattttctttaggagattt 14164
```

Query: 3332 tttggacttcaaagcaagctggtatttcatacaaattcttaattgctgtgtccca 3391
||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||
Sbjct: 14165 tttggacttcaaagcaagctggtatttcatacaaattcttaattgctgtgtccca 14224

Query: 3392 ggcagggagacgggttccaggagggggccggccctgtgtgcaggttccatgttattaga 3451
||||||||||||||||||||||||||||||||||||||||||||||||||||
Sbjct: 14225 ggcagggagacgggttccaggagggggccggccctgtgtgcaggttccatgttattaga 14284

Query: 3452 tgttacaagtt 3462
|||||||||||
Sbjct: 14285 tgttacaagtt 14295

FILE 'MEDLINE, BIOSIS, CANCERLIT, LIFESCI, BIOTECHDS, CAPLUS' ENTERED AT
20:01:05 ON 18 JAN 2003

L1 8 S M64347##

L2 7 DUP REM L1 (1 DUPLICATE REMOVED)

L3 7949 S (FGF(W)R) OR (FGF(W)R3) OR
(FIBROBLAST(W)GROWTH(W)FACTOR(W)(R)

L4 115579 S NEUROCYTOMA# OR PINEALOMA# OR GLIOMA# OR MEDULLOBLASTOMA#
OR

L5 132127 S (CEREBRAL OR (CHOROID(W)PLEXUS) OR CEREBELLUM OR
HYPOTHALAMIC

L6 10081 S (CEREBRAL OR (CHOROID(W)PLEXUS) OR CEREBELLUM OR
HYPOTHALAMIC

L7 204265 S L4 OR L5 OR L6

L8 151 S L3 AND L7

L9 115 S L8 AND PY<2001

L10 136 S L8 AND PY<2002

L11 67 DUP REM L10 (69 DUPLICATES REMOVED)

L12 1822 S (FGFR3 OR (FGF(W)R3) OR (FGF(W)RECEPTOR(W)3) OR
(FIBROBLAST(W

L13 15 S L12 AND L7

L14 6 DUP REM L13 (9 DUPLICATES REMOVED)

L15 31656 S GOLUB?/AU OR LANDER?/AU OR POMEROY?/AU OR TAMAYO?/AU

L16 0 S L15 AND L8

L17 1 S L15 AND L12

L18 10 S L15 AND L3

L19 10 S L17 OR L18

L20 4 DUP REM L19 (6 DUPLICATES REMOVED)

10/066,305

L2 ANSWER 5 OF 7 MEDLINE
ACCESSION NUMBER: 2000408205 MEDLINE
DOCUMENT NUMBER: 20349276 PubMed ID: 10889045
TITLE: Repeat polymorphisms within gene regions: phenotypic and evolutionary implications.
AUTHOR: Wren J D; Forgacs E; Fondon J W 3rd; Pertsemlidis A; Cheng S Y; Gallardo T; Williams R S; Shohet R V; Minna J D; Garner H R
CORPORATE SOURCE: Program in Genetics, Southwestern Graduate School of Biomedical Sciences, Dallas, TX, USA.
CONTRACT NUMBER: P50CA70907 (NCI)
SOURCE: AMERICAN JOURNAL OF HUMAN GENETICS, (2000 Aug) 67 (2) 345-56.
Journal code: 0370475. ISSN: 0002-9297.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-AF013956; GENBANK-AF017789; GENBANK-AF032886; GENBANK-AF042838; GENBANK-AF047437; GENBANK-AF060231; GENBANK-D14838; GENBANK-D83492; GENBANK-D86407; GENBANK-D86550; GENBANK-L08835; GENBANK-M55047; GENBANK-M60052; GENBANK-M60315; GENBANK-**M64347**; GENBANK-R12160; GENBANK-R42196; GENBANK-T47177; GENBANK-T62484; GENBANK-T63962; GENBANK-T70173; GENBANK-U49020; GENBANK-U60325; GENBANK-X55313; GENBANK-X70811; GENBANK-X78261; GENBANK-X82209; GENBANK-Y00285; GENBANK-Y11525; +
ENTRY MONTH: 200008
ENTRY DATE: Entered STN: 20000901
Last Updated on STN: 20030105
Entered Medline: 20000821

AB We have developed an algorithm that predicted 11,265 potentially polymorphic tandem repeats within transcribed sequences. We estimate that 22% (2,207/9,717) of the annotated clusters within UniGene contain at least one potentially polymorphic locus. Our predictions were tested by allelotyping a panel of approximately 30 individuals for 5% of these regions, confirming polymorphism for more than half the loci tested. Our study indicates that tandem-repeat polymorphisms in genes are more common than is generally believed. Approximately 8% of these loci are within coding sequences and, if polymorphic, would result in frameshifts. Our catalogue of putative polymorphic repeats within transcribed sequences comprises a large set of potentially phenotypic or disease-causing loci. In addition, from the anomalous character of the repetitive sequences within unannotated clusters, we also conclude that the UniGene cluster count substantially overestimates the number of genes in the human genome.

We hypothesize that polymorphisms in repeated sequences occur with some baseline distribution, on the basis of repeat homogeneity, size, and sequence composition, and that deviations from that distribution are indicative of the nature of selection pressure at that locus. We find evidence of selective maintenance of the ability of some genes to respond very rapidly, perhaps even on intragenerational timescales, to fluctuating selective pressures.

L2 ANSWER 4 OF 7 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:758742 CAPLUS
DOCUMENT NUMBER: 135:314390
TITLE: Large-scale monitoring of expression patterns of
p53-regulated gene and analysis of p53 gene function
INVENTOR(S): Mack, David H.
PATENT ASSIGNEE(S): Affymetrix, Inc., USA
SOURCE: U.S., 46 pp., Cont.-in-part of Appl. No.
PCT/US98/01206.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6303301	B1	20011016	US 1998-86285	19980529
WO 9830722	A1	19980716	WO 1998-US1206	19980112
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ,				
TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
US 2002028454	A1	20020307	US 2001-836278	20010418
PRIORITY APPLN. INFO.:			US 1997-35327P	P 19970113
			WO 1998-US1206	A2 19980112
			US 1997-49627P	P 19970613
			US 1998-86285	A3 19980529

AB This invention provides methods, compns. and app. for mapping the regulatory relationships of genes by massive parallel monitoring of gene expression. The information obtained can be of use in drug discovery (no data). The method uses high d. oligonucleotide arrays to monitor changes in expression in response to events and stimuli. Very large nos. of gene (>6,500) may be monitored in this method using samples from many tissues and developmental or disease stages. Changes are quantified and a relationship model constructed using LISREL (Linear Structure Relationship) methods. Mutations in up-stream regulatory genes can be detected by monitoring the change in down-stream gene expression. Similarly, the effect of a specific mutation in an up-stream gene is

detd.

by monitoring the down-stream gene expression. In addn., regulatory function of a target gene can be detd. by monitoring the expression of a large no. of down-stream genes. The invention also provides specific embodiments for detecting p53 functional homozygous and heterozygous mutations and for detg. the function of p53 mutations.

REFERENCE COUNT: 46 THERE ARE 46 CITED REFERENCES AVAILABLE FOR
THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE
FORMAT

L2 ANSWER 1 OF 7 CAPLUS COPYRIGHT 2003 ACS
 ACCESSION NUMBER: 2002:241013 CAPLUS
 DOCUMENT NUMBER: 136:277466
 TITLE: Expressed gene sets as markers for specific tumors
 INVENTOR(S): Ramaswamy, Sridhar; Golub, Todd B.; Tamayo, Pablo;
 Angelo, Michael
 PATENT ASSIGNEE(S): Whitehead Institute for Biomedical Research, USA;
 Dana-Farber Cancer Institute, Inc.
 SOURCE: PCT Int. Appl., 715 pp.
 CODEN: PIXXD2
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 4
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002024956	A2	20020328	WO 2001-US29287	20010919
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
WO 2002024956	A2	20020328	WO 2001-XA29287	20010919
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
WO 2002024956	A2	20020328	WO 2001-XB29287	20010919
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
WO 2002024956	A2	20020328	WO 2001-XC29287	20010919
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				

AU 2001092802 A5 20020402 AU 2001-92802 20010919
US 2002110820 A1 20020815 US 2001-955920 20010919
PRIORITY APPLN. INFO.: US 2000-233534P P 20000919
US 2001-278749P P 20010326
WO 2001-US29287 W 20010919

AB Sets of genetic markers for specific tumor classes are described, as well as methods of identifying a biol. sample based on these markers. Total RNA was isolated from .apprx.300 human tumor and normal tissue specimens representing 30 individual classes of tumor or normal tissue, and cDNA produced using established mol. biol. protocols was hybridized to two

high

d. Affymetrix oligonucleotide microarrays (Hu6800FL and Hu35KsubA0). Raw expression data was combined into a master data set contg. the expression values for between 6800 and 16,000 genes expressed by each individual sample. A filter was applied to this data set which only allows those genes expressed at 3-fold above baseline and with an abs. difference in expression value of 100 to pass. By comparing the sets of genes which

are

expressed specifically in one class of tumor (e.g., pancreatic adenocarcinoma) vs. its accompanying normal tissue (e.g., normal pancreas), sets of genes were detd. which are specific to various tumors and their normal tissue counterparts. Also described are diagnostic, prognostic, and therapeutic screening uses for these markers, as well as oligonucleotide arrays comprising these markers. [This abstr. record is one of 4 records for this document necessitated by the large no. of index entries required to fully index the document and publication system constraints.].

L2 ANSWER 2 OF 7 CAPLUS COPYRIGHT 2003 ACS

L2 ANSWER 7 OF 7 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 92147110 MEDLINE
DOCUMENT NUMBER: 92147110 PubMed ID: 1664411
TITLE: A gene encoding a fibroblast growth factor receptor isolated from the Huntington disease gene region of human chromosome 4.
AUTHOR: Thompson L M; Plummer S; Schalling M; Altherr M R; Gusella J F; Housman D E; Wasmuth J J
CORPORATE SOURCE: Department of Biological Chemistry, College of Medicine, University of California, Irvine 92717.
CONTRACT NUMBER: NS25631-04 (NINDS)
SOURCE: GENOMICS, (1991 Dec) 11 (4) 1133-42.
Journal code: 8800135. ISSN: 0888-7543.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-M64347
ENTRY MONTH: 199203
ENTRY DATE: Entered STN: 19920405
Last Updated on STN: 20000303
Entered Medline: 19920313
AB The gene responsible for Huntington disease (HD), an autosomal dominant neurodegenerative disorder, is located near the terminus of the short arm of chromosome 4. Detailed genetic linkage and physical mapping studies have defined a region of approximately 2.5 million basepairs where the disease gene is likely to be located. Efforts to identify the disease gene are now focused on the identification and characterization of expressed genes in this region. Nucleotide sequence analysis of a cDNA clone derived from the HD gene region has revealed that it encodes a member of the fibroblast growth factor subfamily of tyrosine kinase receptors, some members of which are known to be involved in the differentiation and survival of certain cell types within the central nervous system. Histochemical analysis using *in situ* hybridization revealed its expression in many areas of the brain, among them being the caudate and putamen. The nature of this gene, FGFR3, and its map location make it a possible candidate for the HD gene.

L2 ANSWER 6 OF 7 CAPLUS COPYRIGHT 2003 ACS
 ACCESSION NUMBER: 1999:641026 CAPLUS
 DOCUMENT NUMBER: 131:267987
 TITLE: Cancer diagnosis and therapy based on expression
 levels of p53-regulated genes
 INVENTOR(S): Levine, Arnold J.; Murphy, Maureen E.; Mack, David
 H.; Gish, Kurt C.; Tom, Edward Yat Wah
 PATENT ASSIGNEE(S): Affymetrix, Inc., USA; Princeton University
 SOURCE: PCT Int. Appl., 46 pp.
 CODEN: PIXXD2
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 1
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9950456	A1	19991007	WO 1999-US6656	19990326
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 6020135	A	20000201	US 1998-49025	19980327
CA 2324444	AA	19991007	CA 1999-2324444	19990326
AU 9932085	A1	19991018	AU 1999-32085	19990326
EP 1064404	A1	20010103	EP 1999-914184	19990326
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
US 6171798	B1	20010109	US 1999-442039	19991117
US 2001039013	A1	20011108	US 2001-755028	20010108
PRIORITY APPLN. INFO.:			US 1998-49025	A1 19980327
			WO 1999-US6656	W 19990326
			US 1999-442039	A3 19991117

AB Many genes are identified as being p53-regulated which were not heretofore

known to be p53-regulated. This includes both genes whose expression is induced and genes whose expression is repressed by the expression of wild-type p53. The effects of p53 expression on gene expression in E6-1 cells was tested by hybridizing to a chip that contains deoxyoligonucleotide sequences (25-mers) that derived from a database of 6800 known genes or EST sequences. Seventy genes were induced by p53 and 77 were repressed by p53. Monitoring expression of these genes is used

to

provide indications of p53 status in a cell. Such monitoring can also be used to screen for useful anticancer therapeutics, as well as for substances which are carcinogenic. Defects in p53 can be bypassed by supplying p53 induced genes to cells. Defects in p53 can also be bypassed

by supplying antisense constructs to p53-repressed genes.

REFERENCE COUNT: 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE

FORMAT

L11 ANSWER 66 OF 67 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1991:79455 CAPLUS
DOCUMENT NUMBER: 114:79455
TITLE: Suppression of basic fibroblast growth factor
expression by antisense oligodeoxynucleotides
inhibits
the growth of transformed human astrocytes
AUTHOR(S): Morrison, Richard S.
CORPORATE SOURCE: Robert S. Dow Neurol. Sci. Inst., Good Samaritan
Hosp., Portland, OR, 97209, USA
SOURCE: Journal of Biological Chemistry (1991),
266(2), 728-34
CODEN: JBCHA3; ISSN: 0021-9258
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Basic fibroblast growth factor (bFGF) is a heparin-binding protein
expressing potent mitogenic and angiogenic properties. Elevated levels
of
bFGF have recently been described in human **glioma** cell lines.
The high degree of vascularity and invasiveness which characterize human
gliomas suggest that activated expression of bFGF or similar
proteins may be related to the aberrant growth patterns of these tumors.
The influence of endogenous bFGF on **glioma** cell growth in vitro
was evaluated in the present study by downregulating bFGF expression
using
antisense oligonucleotide primers. The addn. of 50 .mu.M bFGF-specific
antisense primer to the human **glioma** cell line SNB-19 resulted
in an 80% inhibition in **glioma** growth. This effect was
saturable and specific. Antisense primers directed to 2 different sites
of bFGF mRNA were effective in suppressing SNB-19 growth, whereas sense
strand primer was ineffective. Furthermore, only the antisense primer
significantly reduced the specific activity of bFGF protein in SNB-19
cell
exts. Neither antisense or sense primers inhibited the growth of
non-transformed human glia. BFGF mRNA was detected in both transformed
and nontransformed human glia by polymerase chain reaction anal.
suggesting that alterations in bFGF isoform content or activity may be
specifically related to abnormal growth control in human **gliomas**

L11 ANSWER 65 OF 67 MEDLINE DUPLICATE 26
ACCESSION NUMBER: 91342665 MEDLINE
DOCUMENT NUMBER: 91342665 PubMed ID: 1652059
TITLE: The human **fibroblast growth factor receptor** genes: a common structural arrangement underlies the mechanisms for generating receptor forms that differ in their third immunoglobulin domain.
AUTHOR: Johnson D E; Lu J; Chen H; Werner S; Williams L T
CORPORATE SOURCE: Howard Hughes Medical Institute, Program of Excellence in Molecular Biology, University of California, San Francisco 94143-0724.
CONTRACT NUMBER: HL-43821 (NHLBI)
SOURCE: MOLECULAR AND CELLULAR BIOLOGY, (1991 Sep) 11 (9) 4627-34.
Journal code: 8109087. ISSN: 0270-7306.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199109
ENTRY DATE: Entered STN: 19911013
Last Updated on STN: 19970203
Entered Medline: 19910920
AB To determine the mechanisms by which multiple forms of fibroblast growth factor (FGF) receptors are generated, we have mapped the arrangement of exons and introns in the human FGF receptor 1 (FGFR 1) gene (flg). We found three alternative exons encoding a portion of the third immunoglobulin (Ig)-like domain of the receptor. One of these alternatives encodes a sequence that is part of a secreted form of FGFR 1. The other two encode sequences that are likely part of transmembrane forms of FGFR 1. One of these forms has not been previously reported in published cDNAs. Also, we have determined the structural organization of a portion of the human FGFR 2 gene (bek) and found a similar arrangement of alternative exons for the third Ig-like domain. The arrangement of these genes suggests that there are conserved mechanisms governing the expression of secreted FGF receptors as well as the expression of at least two distinct membrane-spanning forms of the FGF receptors. The diverse forms appear to be generated by alternative splicing of mRNA and selective use of polyadenylation signals.

L14 ANSWER 6 OF 6 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 97229252 MEDLINE
DOCUMENT NUMBER: 97229252 PubMed ID: 9075249
TITLE: Pediatric **brain tumors** express multiple receptor tyrosine kinases including novel cell adhesion kinases.
AUTHOR: Weiner H L; Rothman M; Miller D C; Ziff E B
CORPORATE SOURCE: Department of Neurosurgery (Pediatric Neurosurgery), New York University Medical Center, NY 10016, USA.
CONTRACT NUMBER: P20 NS31088 (NINDS)
SOURCE: PEDIATRIC NEUROSURGERY, (1996 Aug) 25 (2) 64-71;
discussion 71-2.
PUB. COUNTRY: Journal code: 9114967. ISSN: 1016-2291.
Switzerland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199706
ENTRY DATE: Entered STN: 19970612
Last Updated on STN: 20000303
Entered Medline: 19970603
AB We have used the polymerase chain reaction to clone and characterize growth factor receptor tyrosine kinases (RTKs) expressed in 3 pathologically distinct pediatric **brain tumors**, an anaplastic **ependymoma**, a glioblastoma multiforme and a primitive neuroectodermal tumor (PNET). These neoplasms are presumed to be derived from embryonic neuroepithelial precursor cells of the central nervous system. This cloning demonstrated expression of 24 distinct kinase genes: 16 receptor type kinases and 8 nonreceptor type kinases. The expression of 6 receptors, including Hek2, IRR, Ryk, **FGFR3**, and 2 members of the newly identified cell adhesion kinase receptor family, DDR and TKT, in such tumors has not been reported previously. Northern analysis of mRNA levels revealed DDR expression in 6 of 7 pediatric **brain tumors** including an **ependymoma**, PNET, glioblastoma and **astrocytoma**, and also in an adult pheochromocytoma. Thus, the DDR cell adhesion kinase may be widely expressed in pediatric **brain tumors**. Also, PCR cloning may be an effective procedure for characterizing RTKs in clinical tissue samples and revealing the expression of novel RTK species.

WER 5 OF 6 MEDLINE

DUPLICATE 2

ACCESSION NUMBER: 1999085118 MEDLINE

DOCUMENT NUMBER: 99085118 PubMed ID: 9864407

TITLE: Fibroblast growth factor-9 (glia-activating factor)
stimulates proliferation and production of glial
fibrillary

acidic protein in human **gliomas** either in the
presence or in the absence of the endogenous growth factor
expression.

AUTHOR: Miyagi N; Kato S; Terasaki M; Aoki T; Sugita Y; Yamaguchi
M; Shigemori M; Morimatsu M

CORPORATE SOURCE: Department of Pathology, Kurume University, School of
Medicine, Kurume, Japan.

SOURCE: ONCOLOGY REPORTS, (1999 Jan-Feb) 6 (1) 87-92.
Journal code: 9422756. ISSN: 1021-335X.

PUB. COUNTRY: Greece

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199903

ENTRY DATE: Entered STN: 19990402

Last Updated on STN: 19990402

Entered Medline: 19990322

AB We tested fibroblast growth factor-9 (FGF-9) expression in human
glioma cells (U251MG, T98G, U87MG, KALS-1, NMC-G1) and only NMC-G1
expressed endogenous FGF-9. All cells expressed bFGF and high affinity
receptors for FGFs (FGFR1 and **FGFR3**). Exogenously supplied bFGF
and FGF-9 both showed mitogenic activities in all cells. Neutralizing
antibody against bFGF inhibited the proliferation in U251MG and NMC-G1,
however that against FGF-9 inhibited the proliferation only in NMC-G1.
GFAP expression was stimulated by both FGFs in these cells. FGF-9
potentially regulates proliferation and GFAP expression in human
gliomas either in the presence or in the absence of the endogenous
growth factor expression.

L14 ANSWER 4 OF 6 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:756373 CAPLUS
DOCUMENT NUMBER: 136:51990
TITLE: Expression profiling of **medulloblastoma**:
targets PDGFRA and the RAS/MAPK pathway as therapeutic
for metastatic disease
AUTHOR(S): MacDonald, Tobey J.; Brown, Kevin M.; LaFleur,
Bonnie; Peterson, Katia; Lawlor, Christopher; Chen, Yidong;
Packer, Roger J.; Cogen, Philip; Stephan, Dietrich A.
CORPORATE SOURCE: Center for Cancer and Transplantation Biology,
Children's National Medical Center, Washington, DC,
USA
SOURCE: Nature Genetics (2001), 29(2), 143-152
CODEN: NGENEC; ISSN: 1061-4036
PUBLISHER: Nature America Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Little is known about the genetic regulation of **medulloblastoma** dissemination, but metastatic **medulloblastoma** is highly assocd. with poor outcome. We obtained expression profiles of 23 primary **medulloblastomas** clin. designated as either metastatic (M+) or non-metastatic (M0) and identified 85 genes whose expression differed significantly between classes. Using a class prediction algorithm based on these genes and a leave-one-out approach, we assigned sample class to these tumors (M+ or M0) with 72% accuracy and to four addnl. independent tumors with 100% accuracy. We also assigned the metastatic **medulloblastoma** cell line Daoy to the metastatic class. Notably, platelet-derived growth factor receptor .alpha. (PDGFRA) and members of the downstream RAS/mitogen-activated protein kinase (MAPK) signal transduction pathway are upregulated in M+ tumors. Immunohistochem. validation on an independent set of tumors shows significant overexpression of PDGFRA in M+ tumors compared to M0 tumors. Using in vitro assays, we show that platelet-derived growth factor .alpha. (PDGFA) enhances **medulloblastoma** migration and increases downstream MAP2K1 (MEK1), MAP2K2 (MEK2), MAPK1 (p42 MAPK) and MAPK3 (p44 MAPK) phosphorylation in a dose-dependent manner. Neutralizing antibodies to PDGFRA blocks MAP2K1, MAP2K2 and MAPK1/3 phosphorylation, whereas U0126, a highly specific inhibitor of MAP2K1 and MAP2K2, also blocks MAPK1/3. Both inhibit migration and prevent PDGFA-stimulated migration. These results provide the first insight into the genetic regulation of **medulloblastoma** metastasis and are the first to suggest a role for PDGFRA and the RAS/MAPK signaling pathway in **medulloblastoma** metastasis. Inhibitors of PDGFRA and RAS proteins should therefore be considered for investigation as possible novel therapeutic strategies against **medulloblastoma**.
REFERENCE COUNT: 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE REFORMAT

L14 ANSWER 3 OF 6 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2001:265659 BIOSIS
DOCUMENT NUMBER: PREV200100265659
TITLE: Crystal structure of fibroblast growth factor 9 reveals regions implicated in dimerization and autoinhibition.
AUTHOR(S): Plotnikov, Alexander N.; Eliseenkova, Anna V.; Ibrahimi, Omar A.; Shriver, Zachary; Sasisekharan, Ram; Lemmon, Mark A.; Mohammadi, Moosa (1)
CORPORATE SOURCE: (1) Department of Pharmacology, New York University School of Medicine, New York, NY, 10016 USA
SOURCE: Journal of Biological Chemistry, (February 9, 2001) Vol. 276, No. 6, pp. 4322-4329. print.
ISSN: 0021-9258.
DOCUMENT TYPE: Article
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Fibroblast growth factors (FGFs) constitute a large family of heparin-binding growth factors with diverse biological activities. FGF9 was originally described as glia-activating factor and is expressed in the nervous system as a potent mitogen for glia cells. Unlike most FGFs, FGF9 forms dimers in solution with a K_d of 680 nM. To elucidate the molecular mechanism of FGF9 dimerization, the crystal structure of FGF9 was determined at 2.2 ANG resolution. FGF9 adopts a beta-trefoil fold similar to other FGFs. However, unlike other FGFs, the N- and C-terminal regions outside the beta-trefoil core in FGF9 are ordered and involved in the formation of a 2-fold crystallographic dimer. A significant surface area (>2000 ANG²) is buried in the dimer interface that occludes a major receptor binding site of FGF9. Thus, we propose an autoinhibitory mechanism for FGF9 that is dependent on sequences outside of the beta-trefoil core. Moreover, a model is presented providing a molecular basis for the preferential affinity of FGF9 toward **FGFR3**.

L14 ANSWER 2 OF 6 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 2001416780 MEDLINE
DOCUMENT NUMBER: 21359863 PubMed ID: 11466624
TITLE: Frequency of **fibroblast growth factor receptor 3** mutations in sporadic tumours.
AUTHOR: Sibley K; Stern P; Knowles M A
CORPORATE SOURCE: ICRF Clinical Centre, St. James's University Hospital, Leeds, LS9 7TF, UK.
SOURCE: ONCOGENE, (2001 Jul 19) 20 (32) 4416-8.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200108
ENTRY DATE: Entered STN: 20010813
Last Updated on STN: 20010813
Entered Medline: 20010809
AB Mutations in **FGFR3** have been identified in several tumour types including bladder carcinoma, cervical carcinoma, and multiple myeloma. In bladder carcinoma, we recently identified **FGFR3** mutations in 41% of tumours, making this the most frequently mutated putative oncogene identified in bladder cancer to date. We have now investigated the frequency of **FGFR3** mutation in a panel of 125 tumours and 13 cell lines from various other organs. We analysed the mutation hotspots in exons 7, 10 and 15 by direct DNA sequencing, and found one mutation in exon 7 (S249C) in 1/28 (3.5%) cervical tumours. Mutations were not detected in stomach, rectum, colon, prostate, ovarian, breast, brain, or renal tumours, nor were they found in any of the cell lines included in this study. We conclude that **FGFR3** is commonly mutated in bladder carcinoma and only rarely in cervical carcinoma. Several tumour types appear not to possess any mutations in **FGFR3**, suggesting that these mutations are important only in the development of certain types of tumour.

L20 ANSWER 3 OF 4 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 1999308632 MEDLINE
DOCUMENT NUMBER: 99308632 PubMed ID: 10380925
TITLE: Diverse signaling pathways activated by growth factor receptors induce broadly overlapping, rather than independent, sets of genes.
COMMENT: Comment in: Cell. 1999 Jun 11;97(6):675-8
AUTHOR: Fambrough D; McClure K; Kazlauskas A; **Lander E S**
CORPORATE SOURCE: Whitehead Institute for Biomedical Research, Cambridge, Massachusetts 02142, USA.
SOURCE: CELL, (1999 Jun 11) 97 (6) 727-41.
Journal code: 0413066. ISSN: 0092-8674.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199907
ENTRY DATE: Entered STN: 19990730
Last Updated on STN: 20000303
Entered Medline: 19990722

AB We sought to explore the relationship between receptor tyrosine kinase (RTK) activated signaling pathways and the transcriptional induction of immediate early genes (IEGs). Using global expression monitoring, we identified 66 fibroblast IEGs induced by platelet-derived growth factor beta receptor (PDGFRbeta) signaling. Mutant receptors lacking binding sites for activation of the PLCgamma, PI3K, SHP2, and RasGAP pathways still retain partial ability to induce 64 of these IEGs. Removal of the Grb2-binding site further broadly reduces induction. These results

suggest

that the diverse pathways exert broadly overlapping effects on IEG induction. Interestingly, a mutant receptor that restores the RasGAP-binding site promotes induction of an independent group of genes, normally induced by interferons. Finally, we compare the PDGFRbeta and **fibroblast growth factor receptor 1**; each induces essentially identical IEGs in fibroblasts.

L14 ANSWER 32 OF 33 PCTFULL COPYRIGHT 2003 Univentio
 ACCESSION NUMBER: 2001064882 PCTFULL ED 20020822
 TITLE (ENGLISH): 1983, 52881, 2398, 45449, 50289, AND 52872, G
 PROTEIN-COUPLED RECEPTORS AND USES THEREFOR
 TITLE (FRENCH): RECEPTEURS COUPLES A UNE PROTEINE G, NUMEROOTES 1983,
 52881, 2398, 45449, 50289, ET 52872, ET UTILISATIONS
 CORRESPONDANTES
 INVENTOR(S): GLUCKSMANN, Maria, Alexandra; GALVIN, Katherine, M.;
 SILOS-SANTIAGO, Inmaculada
 PATENT ASSIGNEE(S): MILLENNIUM PHARMACEUTICALS, INC.; GLUCKSMANN, Maria,
 Alexandra; GALVIN, Katherine, M.; SILOS-SANTIAGO,
 Inmaculada
 DOCUMENT TYPE: Patent
 PATENT INFORMATION:

	NUMBER	KIND	DATE
DESIGNATED STATES	WO 2001064882	A2	20010907
	AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW GH GM KE LS MW MZ SD SL SZ TZ UG ZW AM AZ BY KG KZ MD RU TJ TM AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE TR BF BJ CF CG CI CM GA GN GW ML MR NE SN TD TG		
APPLICATION INFO.:	WO 2001-US6543	A	20010228
PRIORITY INFO.:	US 2000-60/186,059		20000229

DETD The subject can be a cancer patient e.g., a patient with **brain cancer**, **bone cancer**, or **prostate cancer**. In other embodiments, the subject is a non-human animal, e.g., an experimental animal, e.g., an aithritic rat niodel of. . .

is a method of evaluating a sample. The method includes providing a sample, e.g., from the subject, and determining a gene **expression profile** of the sample, wherein the profile includes a value representing the level of 1983, 52881, 2398, 45449, 50289 and 52872 expression.

The method can further include comparing the value of the **expression profile** (i.e., multiple values) to a reference value or reference profile. The gene **expression profile** of the sample can be obtained by any of the methods described herein.

1 5 (e.g., by providing a nucleic acid. . . an indication that the subject has or is disposed to having a disorders as described herein. The method can be used to **monitor** a **treatment** for such disorders in a subject. For example, the gene **expression profile** can be determined for a sample from a subject undergoing **treatment**.

L14 ANSWER 22 OF 33 PCTFULL COPYRIGHT 2003 Univentio
ACCESSION NUMBER: 2001083781 PCTFULL ED 20020826
TITLE (ENGLISH): 14094, A NOVEL HUMAN TRYPSIN FAMILY MEMBER AND USES
THEREOF
TITLE (FRENCH): 14094, UN NOUVEAU MEMBRE DANS LA FAMILLE DE LA
TRYPSINE

HUMAINE ET SON UTILISATION
INVENTOR(S): MEYERS, Rachel; MACBETH, Kyle, J.
PATENT ASSIGNEE(S): MILLENNIUM PHARMACEUTICALS, INC.; MEYERS, Rachel;
MACBETH, Kyle, J.

DOCUMENT TYPE: Patent

PATENT INFORMATION:

	NUMBER	KIND	DATE
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	WO 2001083781	A2	20011108
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DESIGNATED STATES AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR
CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL
IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG
MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ
TM TR TT TZ UA UG US UZ VN YU ZA ZW GH GM KE LS MW MZ
SD SL SZ TZ UG ZW AM AZ BY KG KZ MD RU TJ TM AT BE CH
CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE TR BF BJ
CF CG CI CM GA GN GW ML MR NE SN TD TG
WO 2001-US13903 A 20010430
US 2000-60/200,621 20000428
US 2000-09/633,300 20000808

DETD . . . cancerous or pre-cancerous tissue where a 14094 polypeptide or nucleic acid is expressed, e.g., breast, ovarian, colon, liver, lung, kidney, or **brain cancer**.

. . . found in a tissue where a 14094 polypeptide or nucleic acid is expressed, e.g., breast, ovarian, colon, liver, lung, kidney, or **brain cancer**.

. . . cancer is a sarcoma, a carcinoma, or an adenocarcinoma. Preferably, the cancer is a breast, ovarian, colon, lung, liver, kidney, or **brain cancer**.

. . . gastric cancer, esophageal cancer, rectal cancer, pancreatic cancer, ovarian cancer, prostate cancer, uterine cancer, cancer of the head and neck, skin **cancer**, **brain cancer**, squamous cell carcinoma, sebaceous gland carcinoma.

. . . can further include comparing the value or the profile (i.e., multiple values) to a reference value or reference profile. The gene **expression profile** of the sample can be obtained by any of the methods described herein (e.g., by providing a nucleic acid from the sample. . . is an indication that the subject has or is disposed to having a cell proliferative disorder. The method can be used to **monitor a treatment** for a cell proliferative disorder in a subject. For example, the gene **expression profile** can be

determined for a sample from a subject

75 -

undergoing **treatment**. The profile can be compared to a reference profile or to a profile obtained from the subject prior to **treatment** or prior to onset of the disorder (see, e.g., Golub et al. (1999) Science 286:531).

.

context, the effect of one cell type on another cell type in response to a biological stimulus can be

determined, e.g., to **monitor** the effect of cell-cell interaction at the level of gene expression,

In another embodiment, cells are contacted with a **therapeutic** agent. The

expression profile of the cells is determined using the array, and the **expression profile** is compared to the profile of like cells not contacted with the agent.

For

example, the assay

can be used to determine or analyze the molecular basis of an undesirable effect of the

therapeutic agent. If an agent is administered

therapeutically to treat one cell type but has an undesirable effect on another cell type, the invention provides an

assay

to determine. . . .

L8 ANSWER 12 OF 20 MEDLINE
ACCESSION NUMBER: 2001393249 MEDLINE
DOCUMENT NUMBER: 21064203 PubMed ID: 11122874
TITLE: Application of advances in molecular biology to the
treatment of brain tumors.
AUTHOR: Takeshima H; Sawamura Y; Gilbert M R; Van Meir E G
CORPORATE SOURCE: Department of Neurosurgery, Faculty of Medicine, Kagoshima
University, 8-35-1 Sakuraga-oka, Kagoshima 890-8520,
Japan.. m2040k@khosp2.kufm.kagoshima-u.ac.jp
SOURCE: Curr Oncol Rep, (2000 Sep) 2 (5) 425-33. Ref: 56
Journal code: 100888967. ISSN: 1523-3790.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200107
ENTRY DATE: Entered STN: 20010716
Last Updated on STN: 20010716
Entered Medline: 20010712
AB Recent advances in molecular biology have substantially improved our
understanding of the molecular genetics of primary brain neoplasms. Soon
each histopathologic category of glioma will be further divided into
subgroups according to similar genetic background, gene **expression**
profile, and similarity of biologic responses to radiotherapy or
chemotherapy. Identification of key molecules that are specifically
altered in neoplastic cells will provide candidate molecular targets for
tumor **treatment**. Novel **therapeutic** tools for targeting
tumor cells, such as viral vectors for gene **therapy**, have been
created. In the near future, the accumulation of new knowledge in brain
tumor biology and genetics, combined with rational drug design, will
revolutionize the **treatment** of malignant gliomas, which are
among the most lethal human cancers.

L14 ANSWER 3 OF 33
ACCESSION NUMBER:
TITLE (ENGLISH):

PCTFULL COPYRIGHT 2003 Univentio
2002059610 PCTFULL ED 20020809 EW 200231
USING OVEREXPRESSION OF LAMININ ALPHA 4 SUBUNIT AS A
DIAGNOSTIC AND PROGNOSTIC INDICATOR OF MALIGNANT

TUMORS

TITLE (FRENCH):

UTILISATION DE LA SUREXPRESSION DE LA SOUS-UNITE DE
LAMININE ALPHA 4 EN TANT QU'INDICATEUR DIAGNOSTIQUE ET
PRONOSTIQUE DE TUMEURS MALIGNES

INVENTOR(S):

LJUBIMOVA, Julia, Y.; LJUBIMOV, Alexander, V.; BLACK,
Keith, L.

PATENT ASSIGNEE(S):

CEDARS-SINAI MEDICAL CENTER
STEINBERG, Nisan, A., Ph.D.

AGENT:

English

LANGUAGE OF FILING:

English

LANGUAGE OF PUBL.:

English

DOCUMENT TYPE:

Patent

PATENT INFORMATION:

	NUMBER	KIND	DATE
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	WO 2002059610	A2	20020801
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DESIGNATED STATES

AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PH PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG UZ VN YU ZA ZW GH GM KE LS MW MZ SD SL SZ TZ UG ZM ZW AM AZ BY KG KZ MD RU TJ TM AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE TR BF BJ CF CG CI CM GA GN GW ML MR NE SN TD TG

APPLICATION INFO.:

WO 2001-US50292 A 20011219

PRIORITY INFO.:

US 2000-09/741,550 20001219

ABEN Disclosed is a method of diagnosing the presence of a malignant tumor, including a glioma, in a human subject, which involves detecting overexpression of laminin *4 subunit protein or laminin *4-specific mRNA, compared to the expression level in a normal tissue control. Also disclosed are a method of predicting the recurrence of a malignant tumor

in a human subject from whom a malignant tumor has been resected and a method of classifying the grade of a malignant tumor, such as a glial tumor, based on a molecular classification.

ABFR L'invention concerne un procede de diagnostic de la presence de tumeurs malignes, y compris de gliomes, chez l'homme, qui implique la detection de la surexpression de la proteine de sous-unite de laminine α 4

ou d'un ARNm specifique de la laminine α 4, par comparaison avec le niveau d'expression dans un témoin tissulaire normal. L'invention concerne également un procede permettant de prévoir la recurrence d'une tumeur maligne chez un homme ayant subi une resection de tumeur

maligne, ainsi qu'un procede de determination de la classe d'une tumeur maligne, telle qu'une tumeur gliale, sur la base d'une classification moleculaire.

L14 ANSWER 4 OF 33 PCTFULL COPYRIGHT 2003 Univentio
 ACCESSION NUMBER: 2002057457 PCTFULL ED 20020801 EW 200230
 TITLE (ENGLISH): 55562 AND 21617, NOVEL HUMAN PROTEINS AND METHODS OF
 USE THEREOF
 TITLE (FRENCH): 55562 ET 21617, NOUVELLES PROTEINES HUMAINES ET LEURS
 METHODES D'UTILISATION
 INVENTOR(S): MEYERS, Rachel, A.; BANDARU, Rajasekhar
 PATENT ASSIGNEE(S): MILLENNIUM PHARMACEUTICALS, INC., for all designates
 States except US; MEYERS, Rachel, A., for US only;
 BANDARU, Rajasekhar, for US only
 AGENT: COLLAZO, Diana, M.
 LANGUAGE OF FILING: English
 LANGUAGE OF PUBL.: English
 DOCUMENT TYPE: Patent
 PATENT INFORMATION:

	NUMBER	KIND	DATE
DESIGNATED STATES	WO 2002057457	A2	20020725
	AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT RO RU SD SE SG SI SK SL TJ TM TN TR TT TZ UA UG US UZ VN YU ZA ZM ZW GH GM KE LS MW MZ SD SL SZ TZ UG ZM ZW AM AZ BY KG KZ MD RU TJ TM AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE TR BF BJ CF CG CI CM GA GN GQ GW ML MR NE SN TD TG		
APPLICATION INFO.:	WO 2001-US49416	A	20011218
PRIORITY INFO.:	US 2000-60/256,249		20001218
	US 2000-60/256,405		20001218

ABEN The invention provides isolated nucleic acids molecules, designated
 21617 and 55562 nucleic acid molecules, which encode novel
 dehydrogenase

or tetratricopeptide repeat members. The invention also provides
 antisense nucleic acid molecules, recombinant expression vectors
 containing 21617 or 55562 nucleic acid molecules, host cells into which
 the expression vectors have been introduced, and nonhuman transgenic
 animals in which a 21617 or 55562 gene has been introduced or
 disrupted.

The invention still further provides isolated 21617 or 55562 proteins,
 fusion proteins, antigenic peptides and anti-21617 or 55562 antibodies.
 Diagnostics methods utilizing compositions of the invention are also
 provided.

ABFR La presente invention concerne des molecules d'acides nucleiques
 isolees, designees sous le nom de molecules d'acides nucleiques 21617
 et

55562, codant de nouvelles repetitions de la deshydrogenase ou du
 tetratricopeptide. L'invention concerne egalement des molecules
 d'acides nucleiques antisens, des vecteurs d'expression recombinés
 contenant ces molecules d'acides nucleiques 21617 ou 55562, des
 cellules

hotes dans lesquelles ces vecteurs d'expression ont ete introduits et
 des animaux transgeniques non humains dans lesquels le gene 21617 ou
 55562 a ete introduit ou interrompu. L'invention concerne egalement des
 proteines 21617 ou 55562 isolees, des proteines hybrides, des peptides
 antigeniques et des anticorps anti-21617 ou 55562. L'invention concerne
 egalement des methodes diagnostiques utilisant des compositions de la

présente invention.

L2 ANSWER 11 OF 23 SCISEARCH COPYRIGHT 2003 ISI (R)
ACCESSION NUMBER: 2000:49185 SCISEARCH
THE GENUINE ARTICLE: 273BX
TITLE: Cell-type specific expression in the pituitary:
physiology
and gene therapy
AUTHOR: Castro M G (Reprint); Windeatt S; SmithArica J;
Lowenstein
CORPORATE SOURCE: UNIV MANCHESTER, SCH MED, MOL MED UNIT, ROOM I-302,
OXFORD
COUNTRY OF AUTHOR: RD, MANCHESTER M13 9PT, LANCS, ENGLAND (Reprint)
ENGLAND
SOURCE: BIOCHEMICAL SOCIETY TRANSACTIONS, (DEC 1999)
Vol. 27, Part 6, pp. 858-863.
Publisher: PORTLAND PRESS, 59 PORTLAND PLACE, LONDON W1N
3AJ, ENGLAND.
ISSN: 0300-5127.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 49

L2 ANSWER 18 OF 23 SCISEARCH COPYRIGHT 2003 ISI (R)
ACCESSION NUMBER: 1998:946863 SCISEARCH
THE GENUINE ARTICLE: 146TR
TITLE: The cytogenesis and pathogenesis of pituitary adenomas
AUTHOR: Asa S L (Reprint); Ezzat S
CORPORATE SOURCE: MT SINAI HOSP, DEPT PATHOL & LAB MED, 600 UNIV AVE,
TORONTO, ON M5G 1X5, CANADA (Reprint); MT SINAI HOSP,
DEPT MED, TORONTO, ON M5G 1X5, CANADA; UNIV TORONTO, DEPT LAB
MED & PATHOBIOLOGY, TORONTO, ON M5G 1X5, CANADA; UNIV
TORONTO, DEPT MED, TORONTO, ON M5G 1X5, CANADA
COUNTRY OF AUTHOR: CANADA
SOURCE: ENDOCRINE REVIEWS, (DEC 1998) Vol. 19, No. 6,
PP. 798-827.
Publisher: ENDOCRINE SOC, 4350 EAST WEST HIGHWAY SUITE
500, BETHESDA, MD 20814-4110.
ISSN: 0163-769X.
DOCUMENT TYPE: General Review; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 361

L2 ANSWER 16 OF 23 SCISEARCH COPYRIGHT 2003 ISI (R)
ACCESSION NUMBER: 1999:271844 SCISEARCH
THE GENUINE ARTICLE: 182EY
TITLE: The pathology of pituitary tumors
AUTHOR: Asa S L (Reprint)
CORPORATE SOURCE: MT SINAI HOSP, DEPT PATHOL & LAB MED, 600 UNIV AVE,
TORONTO, ON M5G 1X5, CANADA (Reprint); UNIV TORONTO, DEPT
LAB MED & PATHOBIOLOG, TORONTO, ON, CANADA
COUNTRY OF AUTHOR: CANADA
SOURCE: ENDOCRINOLOGY AND METABOLISM CLINICS OF NORTH AMERICA, (MAR 1999) Vol. 28, No. 1, pp. 13-6.
Publisher: W B SAUNDERS CO, INDEPENDENCE SQUARE WEST
CURTIS CENTER, STE 300, PHILADELPHIA, PA 19106-3399.
ISSN: 0889-8529.
DOCUMENT TYPE: General Review; Journal
FILE SEGMENT: LIFE; CLIN
LANGUAGE: English
REFERENCE COUNT: 153
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB The pathologist plays an important role in the distinction of
pituitary
adenomas from other tumors and tumor-like lesions of the sellar region,
and in the accurate morphologic characterization of pituitary adenomas. A
clinicopathologic classification of pituitary adenomas is based on cell
differentiation correlated with clinical evidence of hormone secretion;
this classification emphasizes clinically relevant features that can
offer
guidance for patient management. The application of a rational approach
to
the immunohistochemical analysis of these lesions can be used to evaluate
pathogenetic and prognostic markers and to predict responses to specific
therapeutic modalities.